

Chromosome 10

Description

Humans normally have 46 chromosomes in each cell, divided into 23 pairs. Two copies of chromosome 10, one copy inherited from each parent, form one of the pairs.

Chromosome 10 spans more than 133 million DNA building blocks (base pairs) and represents between 4 and 4.5 percent of the total DNA in cells.

Identifying genes on each chromosome is an active area of genetic research. Because researchers use different approaches to predict the number of genes on each chromosome, the estimated number of genes varies. Chromosome 10 likely contains 700 to 800 genes that provide instructions for making proteins. These proteins perform a variety of different roles in the body.

Health Conditions Related to Chromosomal Changes

The following chromosomal conditions are associated with changes in the structure or number of copies of chromosome 10.

10q26 deletion syndrome

10q26 deletion syndrome is a condition that results from the loss (deletion) of a small piece of chromosome 10 in each cell. The deletion occurs on the long (q) arm of the chromosome at a position designated 10q26.

The signs and symptoms of 10q26 deletion syndrome vary widely, even among affected members of the same family. Affected individuals may have distinctive facial features, growth problems, mild to moderate intellectual disability, developmental delay, genital abnormalities in males, or skeletal or heart defects.

People with 10q26 deletion syndrome are missing between 3.5 million and 17 million DNA building blocks (base pairs), also written as 3.5 and 17 megabases (Mb), at position q26 on chromosome 10. The exact size of the deletion varies, and it is unclear what exact region needs to be deleted to cause the condition. In many affected individuals, the 10q26 deletions include the tip of the q arm of chromosome 10; however, some smaller deletions occur within the arm of the chromosome.

The signs and symptoms of 10q26 deletion syndrome are probably related to the loss of one or more genes in the deleted region. However, it is unclear which missing genes

contribute to the specific features of the disorder.

Cancers

Changes in the number and structure of chromosome 10 are associated with several types of cancer. For example, a loss of all or part of chromosome 10 is often found in brain tumors called gliomas, particularly in aggressive, fast-growing gliomas. The association of cancerous tumors with a loss of chromosome 10 suggests that some genes on this chromosome play critical roles in controlling the growth and division of cells. Without these genes, cells could grow and divide too quickly or in an uncontrolled way, resulting in cancer. Researchers are working to identify the specific genes on chromosome 10 that may be involved in the development and progression of gliomas.

A complex rearrangement (translocation) of genetic material between chromosomes 10 and 11 is associated with several types of blood cancer known as leukemias. This chromosomal abnormality is found only in cancer cells. It fuses part of a specific gene from chromosome 11 (the *KMT2A* gene) with part of another gene from chromosome 10 (the *MLLT10* gene). The abnormal protein produced from this fused gene signals cells to divide without control or order, leading to the development of cancer.

Other chromosomal conditions

Other changes in the number or structure of chromosome 10 can have a variety of effects. Intellectual disability, delayed growth and development, distinctive facial features, and heart defects are common features. Changes to chromosome 10 include an extra piece of the chromosome in each cell (partial trisomy), a missing segment of the chromosome in each cell (partial monosomy), and an abnormal structure called a ring chromosome 10. Ring chromosomes occur when a chromosome breaks in two places and the ends of the chromosome arms fuse together to form a circular structure. Translocations or inversions (breakage of a chromosome in two places) can also lead to extra or missing material from chromosome 10.

Additional Information & Resources

Additional NIH Resources

- National Human Genome Research Institute: Chromosome Abnormalities (<https://www.genome.gov/about-genomics/fact-sheets/Chromosome-Abnormalities-Fact-Sheet>)

Scientific Articles on PubMed

- PubMed (<https://pubmed.ncbi.nlm.nih.gov/?term=%28Chromosomes,+Human,+Pair+10%5BMAJR%5D%29+AND+%28Chromosome+10%5BTI%5D%29+AND+english%5Bla%5D+AND+human%5Bmh%5D+AND+%22last+1800+days%22%5Bdp%5D>)

References

- Deloukas P, Earthrowl ME, Grafham DV, Rubenfield M, French L, Steward CA, SimsSK, Jones MC, Searle S, Scott C, Howe K, Hunt SE, Andrews TD, Gilbert JG, Swarbreck D, Ashurst JL, Taylor A, Battles J, Bird CP, Ainscough R, Almeida JP, Ashwell RI, Ambrose KD, Babbage AK, Bagguley CL, Bailey J, Banerjee R, Bates K, Beasley H, Bray-Allen S, Brown AJ, Brown JY, Burford DC, Burrill W, Burton J, Cahill P, Camire D, Carter NP, Chapman JC, Clark SY, Clarke G, Clee CM, Clegg S, Corby N, Coulson A, Dhami P, Dutta I, Dunn M, Faulkner L, Frankish A, Frankland JA, Garner P, Garnett J, Gribble S, Griffiths C, Grocock R, Gustafson E, Hammond S, Harley JL, Hart E, Heath PD, Ho TP, Hopkins B, Horne J, Howden PJ, Huckle E, Hynds C, Johnson C, Johnson D, Kana A, Kay M, Kimberley AM, Kershaw JK, Kokkinaki M, Laird GK, Lawlor S, Lee HM, Leongamornlert DA, Laird G, Lloyd C, Lloyd DM, Loveland J, Lovell J, McLaren S, McLay KE, McMurray A, Mashreghi-Mohammadi M, Matthews L, Milne S, Nickerson T, Nguyen M, Overton-Larty E, Palmer SA, Pearce AV, Peck AI, Pelan S, Phillimore B, Porter K, Rice CM, Rogosin A, Ross MT, Sarafidou T, Sehra HK, Shownkeen R, Skuce CD, Smith M, Standring L, Sycamore N, Tester J, Thorpe A, Torcasso W, Tracey A, Tromans A, Tsolas J, Wall M, Walsh J, Wang H, Weinstock K, West AP, Willey DL, Whitehead SL, Wilming L, Wray PW, Young L, Chen Y, Lovering RC, Moschonas NK, Siebert R, Fechtel K, Bentley D, Durbin R, Hubbard T, Doucette-Stamm L, Beck S, Smith DR, Rogers J. The DNA sequence and comparative analysis of human chromosome 10. *Nature*. 2004 May 27;429(6990):375-81. doi: 10.1038/nature02462. Citation on PubMed (<http://pubmed.ncbi.nlm.nih.gov/15164054>)
- Deloukas P, French L, Meitinger T, Moschonas NK. Report of the third international workshop on human chromosome 10 mapping and sequencing 1999. *Cytogenet Cell Genet*. 2000;90(1-2):1-12. doi: 10.1159/000015653. No abstract available. Citation on PubMed (<https://pubmed.ncbi.nlm.nih.gov/11060438>)
- Ensembl Human Map View: Chromosome 10 (http://www.ensembl.org/Homo_sapiens/Location/Chromosome?chr=10;r=10:1-133797422)
- Gilbert F. Chromosome 10. *Genet Test*. 2001 Spring;5(1):69-82. doi: 10.1089/109065701750168824. No abstract available. Citation on PubMed (<https://pubmed.ncbi.nlm.nih.gov/11336406>)
- Lin S, Zhou Y, Fang Q, Wu J, Zhang Z, Ji Y, Luo Y. Chromosome 10q26 deletionsyndrome: Two new cases and a review of the literature. *Mol Med Rep*. 2016 Dec;14(6):5134-5140. doi: 10.3892/mmr.2016.5864. Epub 2016 Oct 19. Citation on PubMed (<https://pubmed.ncbi.nlm.nih.gov/27779662>) or Free article on PubMed Central (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5355737/>)
- Meyer C, Schneider B, Jakob S, Strehl S, Attarbaschi A, Schnittger S, Schoch C, Jansen MW, van Dongen JJ, den Boer ML, Pieters R, Ennas MG, Angelucci E, Koehl U, Greil J, Griesinger F, Zur Stadt U, Eckert C, Szczepanski T, Niggli FK, Schafer BW, Kempski H, Brady HJ, Zuna J, Trka J, Nigro LL, Biondi A, Delabesse E, Macintyre E, Stanulla M, Schrappe M, Haas OA, Burmeister T, Dingermann T, Klingebiel T, Marschalek R. The MLL recombinome of acute leukemias. *Leukemia*. 2006 May;20(5):777-84. doi: 10.1038/sj.leu.2404150. Citation on PubMed (<https://pubmed.ncbi.nlm.nih.gov/16511515>)

- Ohgaki H, Kleihues P. Genetic pathways to primary and secondary glioblastoma. Am J Pathol. 2007 May;170(5):1445-53. doi: 10.2353/ajpath.2007.070011. Citation on PubMed (<https://pubmed.ncbi.nlm.nih.gov/17456751>) or Free article on PubMed Central (<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1854940/>)
- Rasheed BK, Wiltshire RN, Bigner SH, Bigner DD. Molecular pathogenesis of malignant gliomas. Curr Opin Oncol. 1999 May;11(3):162-7. doi:10.1097/00001622-199905000-00004. Citation on PubMed (<https://pubmed.ncbi.nlm.nih.gov/10328589>)
- Scigliano S, Gregoire MJ, Schmitt M, Jonveaux PH, LeHeup B. Terminal deletion of the long arm of chromosome 10. Clin Genet. 2004 Apr;65(4):294-8. doi:10.1111/j.1399-0004.2004.00218.x. Citation on PubMed (<https://pubmed.ncbi.nlm.nih.gov/15025722>)
- UCSC Genome Browser: Statistics (<http://genome.cse.ucsc.edu/goldenPath/stats.html>)
- Van Limbergen H, Poppe B, Janssens A, De Bock R, De Paepe A, Noens L, Speleman F. Molecular cytogenetic analysis of 10;11 rearrangements in acute myeloid leukemia. Leukemia. 2002 Mar;16(3):344-51. doi: 10.1038/sj.leu.2402397. Citation on PubMed (<https://pubmed.ncbi.nlm.nih.gov/11896537>)
- Vera-Carbonell A, Lopez-Gonzalez V, Bafalliu JA, Ballesta-Martinez MJ, Fernandez A, Guillen-Navarro E, Lopez-Exposito I. Clinical comparison of 10q26 overlapping deletions: delineating the critical region for urogenital anomalies. Am J Med Genet A. 2015 Apr;167A(4):786-90. doi: 10.1002/ajmg.a.36949. Epub 2015 Feb 5. Citation on PubMed (<https://pubmed.ncbi.nlm.nih.gov/25655674>)

Last updated September 1, 2019